Fall 10-10-2016

Change in Function, Pain and Quality of Life following Structured Nonoperative Treatment in Patients with Degenerative Cervical Myelopathy: A Systematic Review

Ian Andersson
University of Puget Sound

Anna Dembek
University of Puget Sound

Change in Function, Pain and Quality of Life following Structured Nonoperative Treatment in Patients with Degenerative Cervical Myelopathy: A Systematic Review

Lindsay Tetreault, Mohammed F Shamji*, John Rhee, Heidi Prather, Brian K. Kwon, Jeff Wilson, Ian B. Andersson, Anna H. Dembek, Krystle T. Pagarigan, Joseph R. Dettori, Michael G. Fehlings*

Abstract

Study Design. Systematic review.

Objective. To conduct a systematic review of the literature to determine (1) the change in function, pain and quality of life following structured nonoperative treatment for degenerative cervical myelopathy (DCM); (2) the variability of change in function, pain and quality of life following different types of structured nonoperative treatment; (3) the differences in outcomes observed between certain subgroups (i.e., baseline severity score, duration of symptoms); and (4) negative outcomes and harms resulting from structured nonoperative treatment.

Summary of Background Data. The role of structured nonoperative treatment for the management of DCM is not well defined, and surgery is typically recommended as the default treatment option for patients with moderate and severe myelopathy.

Methods. A systematic search was conducted in Embase, PubMed, and the Cochrane Collaboration for articles published between January 1, 1950 and February 9, 2015. Studies were included if they evaluated outcomes following structured nonoperative treatment, including therapeutic exercise, manual therapy, cervical bracing and/or traction. Outcomes of interest were functional status (Japanese Orthopedic Association (JOA), Nurick), pain in upper extremities and neck, quality of life (Neck Disability Index), and/or conversion to surgery. The quality of each study was evaluated using the Newcaste-Ottawa Scale and the strength of the

overall body of evidence was rated using guidelines outlined by the Grading of Recommendation Assessment, Development and Evaluation Working Group (GRADE).

Results. Of the 570 retrieved citations, eight met the inclusion criteria and were included in this review. There is very low evidence to suggest that structured nonoperative treatment for DCM results in a positive or negative change in function, pain and quality of life as evaluated by the JOA score. There is also limited evidence from three studies indicating that early structured nonoperative treatment (duration of symptoms <1 year) may be associated with positive clinical outcomes. There were no studies that directly compared structured nonoperative treatment types and no studies that explored outcomes based on patient subgroups. The rate of conversion to surgery was reported to be between 23-54%.

Conclusion. There is a lack of evidence to determine the role of nonoperative treatment in patients with DCM. However, in the majority of studies, patients did not achieve clinically significant gains in functional status following structured nonoperative treatment. Furthermore, 23-54% of patients subsequently underwent surgical treatment.

INTRODUCTION

Degenerative cervical myelopathy (DCM) is a progressive spine disease and the most common cause of spinal cord dysfunction in adults worldwide.[1,2] It is caused by age-related alterations to the spinal axis, including degeneration of the facet joints, intervertebral discs and/or vertebral bodies; progressive spinal kyphosis; and/or ligamentous aberrations including ossification, calcification and hypertrophy of the spinal ligaments.[3] These anatomical changes lead to the narrowing of the spinal canal and may result in progressive cord compression, neurological deterioration and significantly reduced quality of life.

Early reports on the natural history have defined DCM as a relatively "benign" conditioj in which patients are often stable for long periods of time following symptom onset.[4,5] However, there is increasing evidence to suggest that DCM is a progressive disorder and that myelopathic individuals may experience a gradual stepwise decline in neurological status.[6] A recent systematic review of the literature reported that 20-60% of patients with symptoms of myelopathy deteriorate by at least one point on the Japanese Orthopedic Association score (JOA) three to six years after initial assessment.[7] It is therefore important to recognize early signs and symptoms of myelopathy in order to implement appropriate treatment strategies to minimize functional loss related to pain and neurological impairment.

Surgery has become increasingly recommended as a "first-line" treatment for patients with DCM, as decompression not only effectively halts disease progression but also results in significant gains in functional status and quality of life.[8-12] In contrast, the effectiveness of structured nonoperative treatment in stabilizing or improving DCM symptoms is not well defined, making it difficult to determine the appropriate role of nonoperative treatment in the

management of DCM, particularly in individuals with mild symptoms. As such, the objective of this study is to conduct a systematic review of the literature to address four clinical questions: In adult patients with DCM,

(1) What is the change in function, pain and quality of life following nonoperative treatment?

(2) Does this change in function, pain and quality of life vary depending on type of nonoperative treatment?

(3) Does the change in function, pain and quality of life following nonoperative care differ across subgroups (e.g., myelopathy severity or duration of myelopathy symptoms)?

(4) What are the harms of nonoperative care and what is the percentage of patients who subsequently undergo surgery?

MATERIALS AND METHODS

Electronic Literature Search

We conducted a systematic search in Embase, PubMed, and the Cochrane Collaboration Library for literature published between January 1, 1950 and February 9, 2015 to identify studies that reported the outcomes of structured nonoperative treatment for the management of DCM. "Structured nonoperative treatment" was defined as any non-surgical intervention and included therapeutic exercise, manual therapy, bracing, cervical traction and others. Our search was limited to human studies published in English. Reference lists from the articles produced by the search were reviewed manually to identify additional publications. For clinical questions 1 through 4, we included studies that reported changes in function, pain and/or health-related quality of life following structured non-operative treatment in adult patients (≥18 years of age) diagnosed with DCM due to spondylosis, herniated discs, and/or ossification of the posterior

longitudinal ligament (OPLL). We also included studies which reported the percentage of patients who ultimately were treated surgically following a period of structured nonoperative treatment, as well as studies that stratified subjects based on baseline myelopathy severity. For clinical question 2, we sought to identify studies that explored competing nonoperative interventions for the management of DCM.

Studies were excluded if they (1) included subjects under 18 years of age or patients with myelopathy due to infection, malignancy, acute injury including acute disc herniation, inflammatory arthritis, or trauma; (2) only reported outcomes following surgical intervention; (3) did not state what type of structured nonoperative treatment was performed; (4) did not evaluate outcome using at least one primary outcome measure (JOA, Nurick, conversion to surgery following nonoperative treatment); (5) reported on fewer than 10 subjects; and/or (6) were related to animals or cadavers, or were strictly biomechanical evaluations. Full inclusion and exclusion criteria are provided in Table 1. Two investigators (AHD, IBA) independently reviewed the full texts of potential articles and excluded all studies that did not meet the inclusion criteria, Figure 1. Selection discrepancies were settled through discussion.

Data Extraction

The following data were extracted from each included article: study design; patient characteristics, including mean age, baseline severity score and type of DCM; length and rate of follow-up; type and duration of nonoperative treatment; outcomes assessed; and associations between nonoperative interventions and outcomes (function, pain, quality of life and/or conversion to surgery). We attempted to identify studies with overlapping data to prevent

double-counting. In such cases, we selected the study with the most complete data, largest sample size and greatest follow-up period.

Study Quality and Overall Strength of Body of Literature

Each article was appraised for risk of bias by two reviewers (KTP, JRD) using the modified Newcastle-Ottawa Scale (NOS).[13] Strength of the overall body of evidence for each outcome was determined by guidelines outlined by the Grading of Recommendation Assessment, Development and Evaluation Working Group (GRADE).[14,15] Though the GRADE scale is intended to rate the quality of evidence and strength of recommendation of comparative studies, we adapted the principles for this systematic review to determine the confidence we have in the magnitude of the effect in the change in function, pain, and quality of life from nonoperative treatment.

The overall body of evidence is considered LOW if all studies are observational. The quality of the body of evidence may be upgraded or downgraded depending upon a number of factors. Criteria for downgrading published evidence one or two levels include (1) inconsistency of results, (2) indirectness of evidence, or (3) imprecision of the effect estimates (e.g., wide variance). Alternately, the body of evidence could be upgraded 1 or 2 levels based on (1) large magnitude of effect or (2) dose-response gradient.

A quality level of HIGH indicates high confidence that the true effect lies close to that of the estimate of effect. A MODERATE quality level reflects moderate confidence in the effect estimate; the true effect is likely to be close to the estimate of the effect, but there is a possibility that it is substantially different. A LOW quality estimate represents limited confidence in the effect estimate, and that the true effect may be substantially different from

the estimate of the effect.[15] VERY LOW ratings indicate very little confidence in the effect estimate; the true effect is likely to be substantially different from the estimate of effect. This rating may be used if there is no evidence or if it is not possible to estimate an effect.

Data Analysis

For clinical questions 1, 2 and 3, changes in JOA or modified JOA (mJOA) scores before (at baseline) and after structured nonoperative treatment were reported and summarized. The minimal clinically important difference (MCID) for the JOA has not been established; however, expert opinion indicates a score change ≥2.0 points is considered clinically significant.[16,17] Furthermore, the MCID of the mJOA has been estimated to be between 1 and 2 points.[18] For clinical question 4, a summary table was used to identify the proportion of subjects that received surgical intervention after a period of structured nonoperative treatment**.**

RESULTS

Study Selection

Our initial search yielded 570 citations. Following title, abstract, and full-text review, we identified eight studies that met our inclusion criteria for clinical questions 1, 2, and 3, Figure 1 and Table 2. Five of these studies also addressed clinical question 4 and reported proportions of subjects that subsequently underwent surgical intervention after a period of structured nonoperative treatment. Of the remaining 562 studies, 541 were excluded at title and abstract level as they primarily focused on surgical intervention and did not appropriately evaluate outcome following structured nonoperative treatment. After full-text review, 20 additional studies were excluded for the following reasons: inappropriate study design (n = 5), inclusion of patients with trauma or radiculopathy (n = 3), abstract publication only (n = 2), inappropriate

outcome measures (n = 2), duplicate data (n = 2), surgical treatment only (n = 1), non-cervical condition (n = 1), non-English publication (n = 1), and no description of structured nonoperative intervention (n = 2). A list of excluded studies and full data abstraction tables can be found in the supplemental electronic material.

Summary of Studies and Risk of Bias

Types of structured nonoperative treatment varied across studies and were not well-defined. Treatments included bed rest, cervical traction, cervical immobilization or bracing thermal therapy, physical therapy and/or non-steroidal anti-inflammatory drugs. Outcomes were assessed using a variety of measures such as the JOA, timed 10-meter walking test, Neck Disability Index (NDI) and Activities of Daily Living (ADL). Some studies also reported rates of conversion to surgery following an initial trial of conservative management.

Based on the modified NOS, six studies had “moderately low risk of bias” and two were rated as “moderately high risk of bias.” Significant methodological flaws included high attrition rate (n = 4), selection bias in choosing source population (n = 1) and small sample sizes (n = 3). A detailed critical appraisal of each study can be found in the Supplemental Electronic Material.

What is the change in function, pain and quality of life following structured nonoperative treatment for degenerative cervical myelopathy?

Assessment of JOA or mJOA scores

Six studies reported outcomes following structured nonoperative treatment using change in JOA (n = 5) or mJOA (n = 1) scores from baseline to follow-up, Table 3. Sample sizes ranged from 32 to 90 subjects, with mean baseline severity scores from 11.1 to 14.6 on the JOA/mJOA. Response to treatment was minimal, with change scores ranging from 0 to 2.3.

Only a single study by Matsumoto *et al*[19] reported a mean JOA change score ≥2.0 points at final follow-up (mean: 47 months).

Two additional studies evaluated outcomes using the JOA but did not report change scores. A retrospective cohort study by Nakamura *et al*[20] evaluated changes in motor function of the upper and lower extremities following a variety of structured nonoperative treatments: continuous head-halter traction (n = 2), cervical bracing (n = 19), plaster bed immobility (n = 15) or Crutchfield's skull traction (n = 28). Extremity function was assessed in sixty-four subjects (74% male, mean age: 54 years) using a disability scale from 0 ("severe impairment") to 4 ("no disability") based on JOA scores. At final follow-up (mean: 47 months), 27% (15/56) and 26% (16/61) of patients who received structured nonoperative treatment had "no disability" in the upper and lower extremities, respectively.

In a second retrospective study, Yoshimatsu *et al*[21] investigated symptomatic changes in 69 patients with DCM who elected not to undergo surgery immediately following diagnosis. Myelopathy severity and functional disability were assessed at baseline using the JOA. Of the 69 subjects, 12 refused treatment, 37 underwent "rigorous" nonoperative care, and 20 received non-rigorous care. "Rigorous" treatment consisted of 3 to 4 hours of continuous cervical traction per day for 1-3 months, combined with immobilization by cervical orthosis, exercise therapy, drug therapy, and thermal therapy. A description of non-rigorous intervention was not provided. To evaluate treatment effects, baseline and post-treatment JOA scores were compared and subjects were classified into three groups based on whether they exhibited "improvement", "no change", or "exacerbation of symptoms" at final follow-up (mean: 29 months). Twenty-six percent (15/57) of patients who received structured nonoperative

treatment demonstrated improvements of JOA scores between baseline and follow-up, and only 8% (1/12) of patients who refused structured nonoperative care exhibited gains. In addition, a smaller percentage of patients who received structured nonoperative care experienced "exacerbation of symptoms" based on the JOA (58%; 33/57) than those who refused nonoperative treatment (83%; 10/12). However, the difference between patients receiving structured nonoperative care and those refusing treatment with respect to JOA improvements and exacerbation of symptoms was within the limits of chance.

Does the change in function, pain and quality of life vary depending on treatment type?

No studies directly compared outcomes between different structured nonoperative treatment strategies; however, one study evaluated outcomes based on different treatment "intensities." A retrospective cohort study by Yoshimatsu *et al*[21] investigated symptomatic changes in 69 patients with DCM who received either rigorous or nonrigorous nonoperative treatment. Thirty-eight percent (14/37) of patients receiving rigorous nonoperative treatment reported some improvement, compared with only 6% (2/32) of patients reporting improvement after receiving non-rigorous nonoperative treatment. The proportion of patients with worsening of symptoms was 49% (18/37) and 78% (25/32), respectively.

Does the change in function, pain and quality of life with nonoperative care vary according to subgroups (i.e., myelopathy severity or duration of symptoms)?

Duration of Symptoms

Three studies evaluated the correlation between duration of symptoms prior to structured nonoperative treatment and post-treatment JOA scores.[20,22] Fukui *et al*[22] evaluated changes in functional disability on the JOA score following 2 weeks of cervical traction.

Pretreatment JOA scores for 53 subjects ranged from 6 to 15 with a mean of 11.1 points (3 subjects refused structured nonoperative treatment, n = 50). Fifty-six percent (28/50) of subjects demonstrated post-treatment JOA improvements. In patients with a duration of symptoms less than three months, 80% (12/15) improved by at least one point on the JOA; in contrast, only 46% (16/35) with a duration of symptoms greater than three months exhibited ≥1 point JOA improvement, risk ratio 1.75 (95% CI 1.13 to 2.72). Nakamura *et al*[20] also evaluated whether duration of symptoms is predictive of JOA improvements following structured nonoperative treatment. For subjects with a duration less than 6 months, 30% (3/10) had "no disability" in the upper extremity and 36% (5/14) had "no disability" in the lower extremity following treatment. For subjects with a symptom duration >6 months, a slightly smaller percentage of patients achieved "no disability" in the upper (26%; 12/46) and lower (23%; 11/47) extremities. Although these differences were not statistically significant, the authors indicated that early intervention could result in improved treatment effects following structured nonoperative treatment.

In a retrospective study, Li *et al*[23] reported a significant correlation between JOA recovery ratios and disease durations ($R=0.888$, $P<0.01$) for a combined surgical and nonoperative group. Patients with a shorter duration of symptoms achieved superior clinical outcomes.

Baseline Severity Score

No studies stratified their sample based on preoperative myelopathy severity.

Other Subgroups

A retrospective cohort study by Matsumoto *et al*[19] evaluated structured nonoperative treatment in patients with myelopathy secondary to cervical soft disc herniation. This study analyzed data from 27 subjects with moderate myelopathy (mean baseline JOA 13.8) who underwent cervical bracing, traction, and NSAID therapy for 6 months with a mean follow-up time of 3.9 years. Sixty-three percent (17/27) of patients demonstrated improvement or stability on the JOA at final follow-up and 59% (10/17) displayed spontaneous regression of their disc herniation and reduction in myelopathy symptoms. The authors concluded that structured nonoperative treatment may reduce neurological symptoms in patients with myelopathy secondary to cervical disc herniation.

What are the harms of nonoperative care and what is the percent of patients who convert to surgery?

No studies reported direct harms resulting from structured nonoperative treatment. Based on five studies, the proportion of subjects who underwent surgical intervention following a period of structured nonoperative treatment ranged from 23% to 54% (mean follow-up: 27 to 74 months), Table 4. In patients with baseline JOA scores ≥13.0, 23% to 38% of patients ultimately received surgery. In patients with more severe myelopathy (JOA<13.0 (11.1)), Fukui *et al*[22] reported a rate of conversion of 54% (27/50) following a period of structured nonoperative treatment. Nakamura *et al*[20] did not specify baseline JOA scores, but did indicate that 30% (19/64) eventually received surgical intervention at a follow up period ranging from 36 to 129 months.

Evidence summary

Eight small studies, ranging in size from 27 to 90 patients, evaluated outcomes following structured nonoperative treatment in patients presenting with mostly mild to moderate DCM (mean baseline mJOA/JOA score ≥12). mJOA or JOA improvement from baseline was generally below the MCID with mean change scores ranging from 0 to 1 in most studies. One subgroup of patients with DCM resulting from soft disc herniation reported 63% (17/27) of patients with improved JOA scores at an average 4 year follow-up. The proportion of patients receiving surgery following nonoperative care ranged from 23% to 54% across five small studies. The quality of evidence for these findings is VERY LOW.

DISCUSSION

There is increasing evidence to support that surgery results in significant and clinically meaningful improvements in functional status and quality of life in patients with varying degrees of myelopathy severity.[8-12] On the other hand, the role of nonoperative treatment in these patients has not been well defined. It was therefore the objective of this review to evaluate changes in function, pain and quality of life outcomes in patients undergoing structured nonoperative treatment for DCM.

We found a lack of evidence to determine the appropriate role of nonoperative treatment in patients with DCM. Furthermore, based on the included studies in this review, the baseline mJOA or JOA either stayed the same or slightly improved following structured nonoperative treatment (0 to 2.3 points). However, there is very low evidence to suggest that these patients exhibit clinically meaningful gains in functional status. In this regard, it is important to consider patient choice as some patients may be satisfied with simply maintaining their level of function whereas others may seek surgical consultation. Furthermore, some

patients may not be ideal surgical candidates due to advanced age or multiple medical co-morbidities.

Interestingly, the greatest reported improvements with nonoperative care occurred in studies involving patients with myelopathy due to soft disc herniation (Matsumoto, diff 2.3) and dynamic cervical myelopathy (Fukui, diff 1.7). These etiologies might be expected *a priori* to respond better to nonoperative care, since soft disc herniations may spontaneously regress, and immobilization may at least temporarily decrease cord irritation if the primary mechanism of compression is dynamic rather than static. In contrast, nonoperative treatment had less effect in studies involving DCM due to static spinal cord compression, or etiologies which do not tend to regress spontaneously over time (Table 3; difference in mJOA/JOA for these studies was 0 to 1.1). Therefore, nonoperative care, based on the evidence in this review, may be reserved for milder myelopathy associated with soft disc herniations or dynamic stenosis.

This review also reported that 23% to 54% of patients converted to surgery following an initial period of conservative treatment. Given this wide range, it is important to predict which patients are at a high risk for disease progression and those who are most likely to eventually undergo surgical intervention. Important predictors of neurological deterioration and failed nonoperative treatment include (1) circumferential cord compression on an axial magnetic resonance image;[24] (2) an "angular-edged" spinal cord, defined as an acute angled or lateral corner at one or both sides;[25] (3) greater range of preoperative neck and head motion;[26] (4) lower segmental lordotic angle and greater percentage of vertebral slip;[27] and (5) segmental instability and reduced diameter of the cerebrospinal fluid column.[28] For patients who are in these high-risk groups, early surgery may be considered regardless of their myelopathy severity.

This is especially critical given recent reports that a longer duration of preoperative symptoms is predictive of a worse surgical outcome.[29,30]

Results of nonoperative management need to be separately evaluated in patients with varying myelopathy severities to better define its role. In a systematic review by Rhee et al (2013), the comparative effectiveness of surgery versus nonoperative management was explored.[31] This review reported that there is little evidence to suggest that nonoperative treatment halts or reverses the progression of myelopathy and that nonoperative care should not be the primary treatment modality in patients with moderate to severe disease. Therefore, surgery should be considered in those with moderate to severe symptoms s without significant delay, as further disease progression could result in considerable harm, reduced quality of life, significant functional disability, and decreased responsiveness to surgery. In addition, Wu et al. found that myelopathic patients may be at a higher risk of sustaining a spinal cord injury or experiencing central cord syndrome following a fall, both of which are associated with not only individual neurologic but also societal economic burdens due to significant increased costs of management.[32]

There may be a potential role for nonoperative management in patients with milder and stable disease forms. Because no studies stratified their samples into cohorts based on preoperative severity, we are unable to determine whether patients with mild myelopathy (mJOA≥15) improve by the MCID on the mJOA/JOA following structured nonoperative treatment.

Limitations

Clinicians who treat myelopathic patients may ask the question *"is it reasonable to prescribe an initial trial of nonoperative care for patients with DCM?"* This systematic review reveals significant flaws in the literature and cannot provide a strong evidence-based answer to this question. The major limitation in the body of evidence is that the type of "structured nonoperative care" is often poorly defined and consists a myriad of treatments, including traction, bracing, massage, exercise and drug administration. The variability of treatment modalities across studies makes it challenging to derive conclusions regarding the effectiveness of nonoperative care for DCM. As seen in Table 5, the level of evidence for each question was deemed "VERY LOW," reflecting little confidence that the estimation of the treatment effect matches the true effect.

There are additional limitations in the body of the evidence. Studies included in this review poorly defined treatment parameters. For example, four studies reported that drug therapy was used as a method of structured nonoperative care.[19,21,23,34] However, none of these studies defined the types of drugs, dosing instructions, or duration of use. Additionally, three studies used other forms of treatment including exercise, thermal therapy or physical therapy[19,21,23] but did not provide further description of these treatments, whether they overlapped, how intensely they were administered, and how compliant individuals were. As a result, we are unable to draw concrete conclusions about the superiority of various conservative treatment modalities over other strategies.

Second, although most studies evaluated functional status using the JOA, one study used the mJOA, a scale developed to account for cultural differences between Eastern and Western societies.[33,34] A recent study by Kato *et al*[16] compared the original JOA with the mJOA

and determined that, although the two scales are highly correlated (Spearman's rho = 0.87), it is not ideal to use them interchangeably. Consequently, the ability to generalize mJOA data with JOA data is limited. Furthermore, two studies used different methods to assess functional status that could not be fully compared to change in JOA or mJOA scores.

Third, the MCID of the mJOA has been shown to vary depending on myelopathy severity: 1 in mild patients (mJOA≥15), 2 in moderate patients (mJOA=12-14) and 3 in severe patients (mJOA<12).[18] However, the studies included in this review did not stratify their sample based on preoperative severity scores. There may be a role for nonoperative treatment in mild patients (mJOA≥15) if they could demonstrate improvements on the mJOA by 1 or more points.

Finally, there is a wide range of follow-up duration among the included studies, which makes it difficult to discern changes due to intervention from changes due to natural disease progression.

Figure 1. Results of literature search

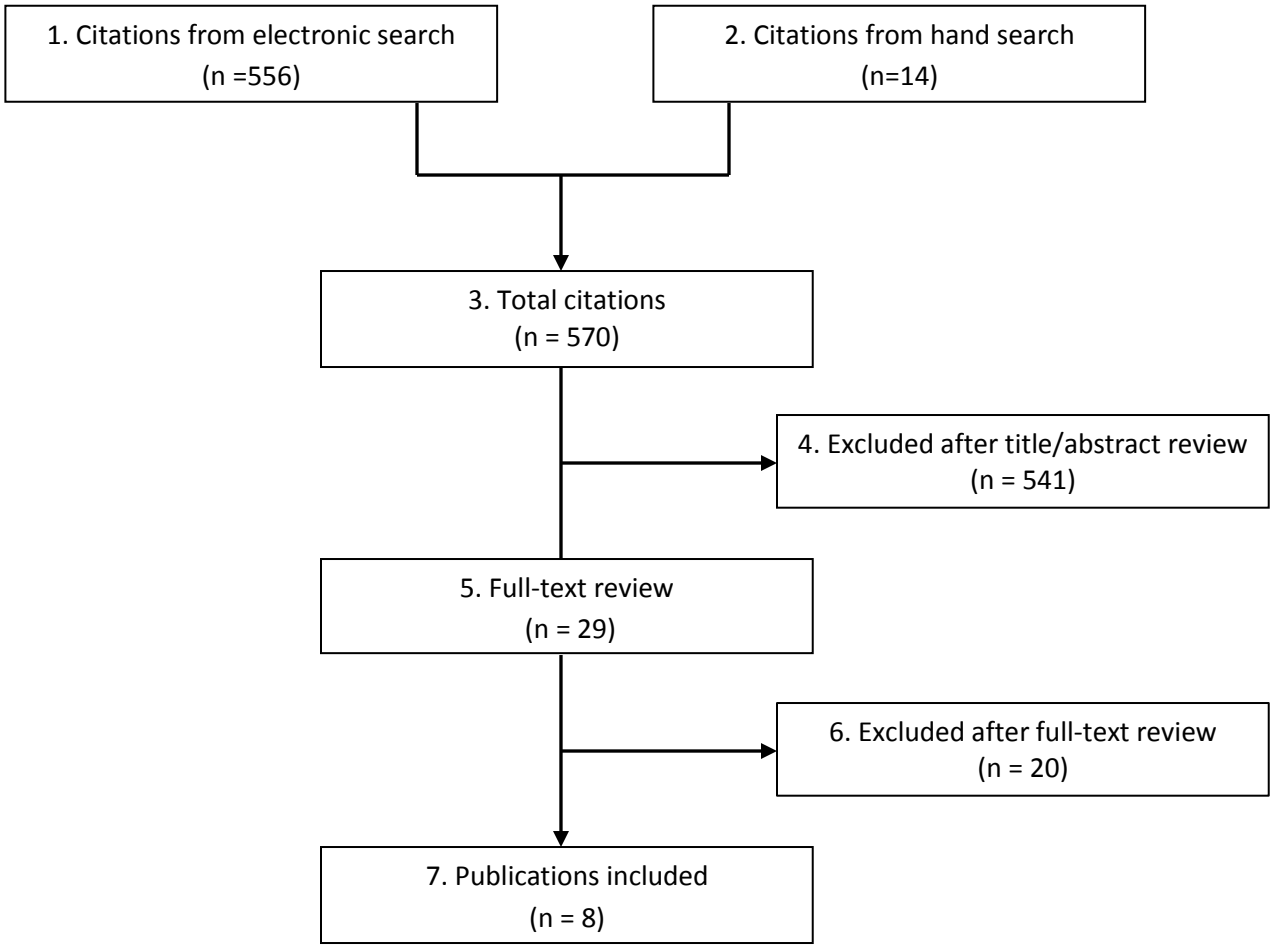

Table 1. PICO Table

	Inclusion:	**Exclusion:**
Patient	Clinical Questions 1-4: • Adult patients (≥ 18 years) with diagnosed myelopathy due to spondylosis, herniated disc, and/or ossification of the posterior longitudinal ligament.	• Patients < 18 years of age • Myelopathy due to infection, malignancy, acute injury, inflammatory arthritis or trauma
Intervention	Clinical Questions 1-4: • Therapeutic exercise • Manual therapy • Bracing • Cervical traction • Other nonoperative treatments	• Surgical intervention
Comparison	Clinical Question 2: • Competing non-operative intervention	
Outcomes	Clinical Questions 1-4: • Quality of life (SF-36 NDI) • Pain (VAS) • Functional status (JOA, Nurick) • Follow-up interview (progression and management) • Conversion to surgery	
Study Design	Clinical Question 2: • RCT • Cohort Studies Clinical Questions 1,3,4: • Case series	• Case reports • Literature review • Narrative review • Animal studies • Studies with <10 patiets

SF-36 indicates Short-Form 36; NDI: Neck Disability Index; VAS, Visual Analog Scale; JOA, Japanese Orthopedic Association; RCT, Randomized Controlled Trials

Table 2. Characteristics of studies

Author(Years) Study Design	Patient Characteristics	Condition, Severity, Duration	Intervention	Outcome measures	Mean f/u (range) % f/u	Risk of Bias
Fukui *et al* (1990)[22] Prospective cohort	N=50* Mean age: 58.6 79% male	Condition: DCM 2° to dynamic canal stenosis Baseline JOA: 11.1 Mean Sx† duration: 2 years, 11 months; range, 1 month to 10 years	Continuous cervical traction 2 weeks (n=50)	JOA score Surgery	2.5 yrs (range NR) %NR	High Risk
Kadanka *et al* (2002; 2011)[33,34] RCT ‡	N=35 Mean age: 54 74.3% male	Condition: DCM Baseline mJOA: 14.6 Median Sx duration: 1 year; range, 0.3 to 6 years	Intermittent cervical bracing with soft collar (n=NR) NSAIDs (n=NR) Intermittent bed rest 2° to pain (n=NR)	Modified JOA score Time 10-m walk ADL score Subjective assessment	12 mos: NR 24 mos: NR 36 mos: 83% 120 mos: 78%	Moderately High Risk
Kong *et al* (2013) Prospective cohort	N=90 Mean age: 57.8 58% male	Condition: DCM Baseline JOA: 14.2 ±1.0 Mean Sx duration: 20.3 months	Continuous cervical traction 8 hours/day for 2 weeks (Good-Samaritan) (n=90)	JOA score Surgery	40 months (36-56 mos) 87%	Low Risk
Li *et al* (2014)[23] Retrospective cohort	N=38§ Mean age: 51.7 52% male	Condition: CSM Baseline JOA: 14.4 Mean Sx duration: 5.97±5.08 months	Oral drugs (n=NR) Traction (n=NR) Acupuncture (n=NR) Physiotherapy (n=NR) Other conservative treatments (n=NR)	JOA score NDI	30.7 mos (range NR) % NR	High Risk
Matsumoto *et al* (2001)[19] Retrospective cohort	N=27 Mean age: 44.4 74% male	Condition: DCM 2° to Soft Disc Herniation Baseline JOA: 13.8 ±1.6	Cervical bracing 8 hours/day for 3 months (n=17) Physical therapy with intermittent cervical traction (n=4) NSAIDs (n=7)	JOA Surgery	3.9 yrs (1-7 yrs) %NR	High Risk

Author(Years) Study Design	Patient Characteristics	Condition, Severity, Duration	Intervention	Outcome measures	Mean f/u (range) % f/u	Risk of Bias
		Mean Sx duration: 4.7 months				
Nakamura *et al* (1998)[20] Retrospective cohort	N=64 Mean age: 52 72% male	Condition: DCM Baseline JOA: NR Mean Sx duration: 24 months; range, 1 month to 20 years	Continuous head halter traction (n=2) Cervical bracing (n=19) Plaster bed immobilization (n=15) Crutchfield skull traction (n=28)	JOA Surgery	74 mos (3-10 yrs) 83%**	Moderately High Risk
Shimomura *et al* (2007) Prospective cohort	N=70 Mean age: 55.1 54% male	Condition: DCM Baseline JOA: 14.6 ±1.3 Sx duration: NR	Continuous cervical traction 8 hours/day for 2 weeks (Good-Samaritan) (n=70)	JOA	35.6 mos (10-60 mos) 80%	Moderately Low Risk
Yoshimatsu et al (2001)[21] Retrospective cohort	N=57†† Mean age: 67 51% male	Condition: DCM Baseline JOA: NR Mean Sx duration: 28.5 months	Continuous cervical traction 3-4 hours/day for 1-3 months (Good-Samaritan) (n=NR) Immobilization (n=NR) Drug therapy (n=NR) Exercise therapy (n=NR) Thermal therapy (n=NR)	JOA Surgery	29 mos (1-76 mos) NR	Moderately High Risk

f/u, follow-up; CSM, Cervical Spondylotic Myelopathy; JOA, Japanese Orthopaedic Association; NR, Not Reported; ADL, activities of daily living; NSAIDS, non-steroidal anti-inflammatory drugs; NDI, Neck Disability Index; OPLL, ossification of the posterior longitudinal ligament; RCT, randomized controlled trial

* N=53; 3 subjects refused conservative treatment

† Sx = symptom

‡ RCT by design; however, data only extracted from conservative arm (prospective cohort)

§ N=91; n=38 in conservative arm

** 19 subjects with surgical outcome and 34 with continued conservative treatment; total of 53 subjects remained for final follow-up (83%)

†† N=101 conservative and surgical arms; 12 subjects in the original conservative arm (n=69) refused treatment

Table 3. Change in (modified) Japanese Orthopedic Association score following conservative treatment in patients with degenerative cervical myelopathy

				JOA*		
Author	**N**	**F/U (mo)**	**Treatment†**	**Baseline**	**Post**	**Diff**
Kadanka *et al* 2002/2011[33,34]	32	36, 120	Immobilization	14.6	14.7	0.1
Li *et al* 2014[23]	38	30.7	Mixed	14.4	15.5	1.1
Matsumoto *et al* 2001[19]	27	47 (12-84)	Mixed	13.8 ±1.6	16.1 ±0.9	2.3
Fukui *et al* 1990[22]	50	30	Traction	11.1	12.8	1.7
Shimomura *et al* 2007[24]	70	35.6 (10-60)	Traction	14.6 ±1.3	14.7 ±2.0	0.1
Kong et al 2013[28]	90	40 (36-56)	Traction	14.2 ±1.0	14.2 ±1.3	0

* 17-point JOA used in all studies except Kadanka *et al*, who used the 18 point modified JOA

† See Table 1 for treatment details

Table 4. The proportion of patients with degenerative cervical myelopathy requiring surgery following conservative treatment.

Author	**N**	**F/U (mo)**	**Treatment***	**Baseline JOA**	**n (%)**
Matsumoto *et al* 2001[19]	27	47 (12-84)	Mixed	13.8 ±1.6	10 (37%)
Fukui *et al* 1990[22]	50	30	Traction	11.1	27 (54%)
Kong *et al* 2013[28]	90	40 (36-56)	Traction	14.2 ±1.0	21 (23%)
Nakamura *et al* 1998[20]	64	74 (36-129)	Mixed	NR	19 (30%)
Yoshimatsu *et al* 2001[21]	57	29 (1-76)	Mixed	NR	22 (39%)

* See Table 1 for treatment details

Table 5. GRADE Summary Table

	Number of Studies (N)	Strength of Evidence	Conclusions
Clinical Question 1: What is the change in function, pain and quality of life following structured nonoperative treatment?			
mJOA/JOA improvement	4 prospective cohorts, 4 retrospective cohorts (n=491)	VERY LOW	There were no clinical or statistically significant differences between mJOA/JOA scores at baseline and follow-up following structured nonoperative treatment for DCM. Evidence was inconsistent across studies: follow-up time ranged from 30 to 74.0 months, baseline mJOA/JOA score ranged from 11.1 to 14.6 points, and change in post-treatment scores ranged from -0.7 to 2.3. One study reported improvement in JOA score in 18% of their patient population.
Clinical Question 2: Does the change in function, pain and quality of life depend on treatment type?			
% of patients with JOA improvement	1 retrospective cohort (n=57)	VERY LOW	1 study reported on the proportion of patients improving by ≥1 on the JOA score following "rigorous" versus "non-rigorous" structured nonoperative treatment. The "non-rigorous" treatment type was not defined.
Clinical Question 3: Does the change in function, pain and quality of life following nonoperative care differ across subgroups?			
Duration: **≤ 3 vs. > 3 months** JOA: ≥ 1 pt. improvement **< 6 vs. ≥ 6 months** **UE JOA: any improvement** **LE JOA: any improvement**	1 prospective cohort (n=50) 1 retrospective cohort (n=61)	VERY LOW	≤ 3 mos: 80% > 3 mos: 46% p=.033 < 6 mos: UE: 30%; LE: 36% ≥ 6 mos: UE: 26%; LE: 23% p=ns for UE & LE
Soft disc herniation **JOA score**	1 retrospective cohort (n=27)	VERY LOW	63% improved
Clinical Question 4: What are the negative patient outcomes and harms?			
Surgery following nonoperative care	2 prospective cohorts, 3 retrospective cohorts (n=288)	VERY LOW	5 studies reported proportion of subjects converting to surgery following a period of structured nonoperative treatment. 23-54% of patients received surgery following structured nonoperative treatment in mostly mild to moderate DCM cases.

References

1. Kalsi-Ryan S, Karadimas SK, Fehlings MG. Cervical spondylotic myelopathy: the clinical phenomenon and the current pathobiology of an increasingly prevalent and devastating disorder. *The Neuroscientist : a review journal bringing neurobiology, neurology and psychiatry.* 2013;19(4):409-421.
2. Tracy JA, Bartleson JD. Cervical spondylotic myelopathy. *The neurologist.* 2010;16(3):176-187.
3. Nouri A, Tetreault L, Singh A, Karadimas SK, Fehlings MG. Degenerative Cervical Myelopathy: Epidemiology, Genetics, and Pathogenesis. *Spine.* 2015;40(12):E675-693.
4. Lees F, Turner JW. Natural History and Prognosis of Cervical Spondylosis. *British medical journal.* 1963;2(5373):1607-1610.
5. Nurick S. The natural history and the results of surgical treatment of the spinal cord disorder associated with cervical spondylosis. *Brain : a journal of neurology.* 1972;95(1):101-108.
6. Matz PG, Anderson PA, Holly LT, et al. The natural history of cervical spondylotic myelopathy. *Journal of neurosurgery. Spine.* 2009;11(2):104-111.
7. Karadimas SK, Erwin WM, Ely CG, Dettori JR, Fehlings MG. Pathophysiology and natural history of cervical spondylotic myelopathy. *Spine.* 2013;38(22 Suppl 1):S21-36.
8. Fehlings MG, Wilson JR, Kopjar B, et al. Efficacy and safety of surgical decompression in patients with cervical spondylotic myelopathy: results of the AOSpine North America prospective multi-center study. *The Journal of bone and joint surgery. American volume.* 2013;95(18):1651-1658.
9. Fehlings MG, Ibrahim A, Tetreault L, et al. A Global Perspective on the Outcomes of Surgical Decompression in Patients with Cervical Spondylotic Myelopathy: Results from the Prospective Multicenter AOSpine International Study on 479 patients. *Spine.* 2015.
10. Cheung WY, Arvinte D, Wong YW, Luk KD, Cheung KM. Neurological recovery after surgical decompression in patients with cervical spondylotic myelopathy - a prospective study. *International orthopaedics.* 2008;32(2):273-278.

11. Gok B, Sciubba DM, McLoughlin GS, et al. Surgical treatment of cervical spondylotic myelopathy with anterior compression: a review of 67 cases. *Journal of neurosurgery. Spine.* 2008;9(2):152-157.
12. Chiles BW, 3rd, Leonard MA, Choudhri HF, Cooper PR. Cervical spondylotic myelopathy: patterns of neurological deficit and recovery after anterior cervical decompression. *Neurosurgery.* 1999;44(4):762-769; discussion 769-770.
13. Wells GA SB, O'Connell D, Peterson J, Welch V, Losos M, Tugwell P. The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses.
14. Atkins D, Best D, Briss PA, et al. Grading quality of evidence and strength of recommendations. *BMJ (Clinical research ed.).* 2004;328(7454):1490.
15. Balshem H, Helfand M, Schunemann HJ, et al. GRADE guidelines: 3. Rating the quality of evidence. *Journal of clinical epidemiology.* 2011;64(4):401-406.
16. Kato S, Oshima Y, Oka H, et al. Comparison of the Japanese Orthopaedic Association (JOA) Score and Modified JOA (mJOA) Score for the Assessment of Cervical Myelopathy: A Multicenter Observational Study. *PloS one.* 2015;10(4):e0123022.
17. Furlan JC, Kalsi-Ryan S, Kailaya-Vasan A, Massicotte EM, Fehlings MG. Functional and clinical outcomes following surgical treatment in patients with cervical spondylotic myelopathy: a prospective study of 81 cases. *Journal of neurosurgery. Spine.* 2011;14(3):348-355.
18. Tetreault L, Kopjar B, Cote P, Nouri A, Fehlings M. The Minimum Clinically Important Difference of the modified Japanese Orthopedic Association Scale in Patients with Degenerative Cervical Myelopathy. *Spine.*
19. Matsumoto M, Chiba K, Ishikawa M, Maruiwa H, Fujimura Y, Toyama Y. Relationships between outcomes of conservative treatment and magnetic resonance imaging findings in patients with mild cervical myelopathy caused by soft disc herniations. *Spine (Phila Pa 1976).* 2001;26(14):1592-1598.
20. Nakamura K, Kurokawa T, Hoshino Y, Saita K, Takeshita K, Kawaguchi H. Conservative treatment for cervical spondylotic myelopathy: achievement and sustainability of a level of "no disability". *J Spinal Disord.* 1998;11(2):175-179.

21. Yoshimatsu H, Nagata K, Goto H, et al. Conservative treatment for cervical spondylotic myelopathy. prediction of treatment effects by multivariate analysis. *The spine journal : official journal of the North American Spine Society.* 2001;1(4):269-273.
22. Fukui K, Kataoka O, Sho T, Sumi M. Pathomechanism, pathogenesis, and results of treatment in cervical spondylotic myelopathy caused by dynamic canal stenosis. *Spine.* 1990;15(11):1148-1152.
23. Li FN, Li ZH, Huang X, et al. The treatment of mild cervical spondylotic myelopathy with increased signal intensity on T2-weighted magnetic resonance imaging. *Spinal cord.* 2014;52(5):348-353.
24. Shimomura T, Sumi M, Nishida K, et al. Prognostic factors for deterioration of patients with cervical spondylotic myelopathy after nonsurgical treatment. *Spine.* 2007;32(22):2474-2479.
25. Sumi M, Miyamoto H, Suzuki T, Kaneyama S, Kanatani T, Uno K. Prospective cohort study of mild cervical spondylotic myelopathy without surgical treatment. *Journal of neurosurgery. Spine.* 2012;16(1):8-14.
26. Barnes MP, Saunders M. The effect of cervical mobility on the natural history of cervical spondylotic myelopathy. *Journal of neurology, neurosurgery, and psychiatry.* 1984;47(1):17-20.
27. Oshima Y, Seichi A, Takeshita K, et al. Natural course and prognostic factors in patients with mild cervical spondylotic myelopathy with increased signal intensity on T2-weighted magnetic resonance imaging. *Spine.* 2012;37(22):1909-1913.
28. Kong LD, Meng LC, Wang LF, Shen Y, Wang P, Shang ZK. Evaluation of conservative treatment and timing of surgical intervention for mild forms of cervical spondylotic myelopathy. *Experimental and Therapeutic Medicine.* 2013;6(3):852-856.
29. Tetreault LA, Kopjar B, Vaccaro A, et al. A clinical prediction model to determine outcomes in patients with cervical spondylotic myelopathy undergoing surgical treatment: data from the prospective, multi-center AOSpine North America study. *The Journal of bone and joint surgery. American volume.* 2013;95(18):1659-1666.
30. Tetreault LA, Karpova A, Fehlings MG. Predictors of outcome in patients with degenerative cervical spondylotic myelopathy undergoing surgical treatment: results of a systematic review. *European spine journal : official publication of the European Spine*

Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society. 2015;24 Suppl 2:236-251.

31. Rhee JM, Shamji MF, Erwin WM, et al. Nonoperative management of cervical myelopathy: a systematic review. *Spine.* 2013;38(22 Suppl 1):S55-67.
32. Wu JC, Ko CC, Yen YS, et al. Epidemiology of cervical spondylotic myelopathy and its risk of causing spinal cord injury: a national cohort study. *Neurosurgical focus.* 2013;35(1):E10.
33. Kadanka Z, Bednarik J, Novotny O, Urbanek I, Dusek L. Cervical spondylotic myelopathy: conservative versus surgical treatment after 10 years. *Eur Spine J.* 2011;20(9):1533-1538.
34. Kadanka Z, Mares M, Bednanik J, et al. Approaches to spondylotic cervical myelopathy: conservative versus surgical results in a 3-year follow-up study. *Spine.* 2002;27(20):2205-2210; discussion 2210-2201.

Supplemental Electronic (SE) Material

SE Table A. Excluded Studies

Author	Year	Reason for exclusion
Arnasson, O.	1987	Myelopathy group treated with surgery only
Boyce, R.H.	2003	Review article
Braakman, R.	1994	Review article
Browder, D.A.	2004	Small sample (n=7); 2 subjects with trauma
Endo, S.	1994	All trauma subjects
Fehlings, M.G.	2013	Consensus statement
Kadanka, Z.	2000	Duplicate of included study (2011)
Kadanka, Z.	2005	Duplicate of 2011 data
Law, M.D.	1995	Review article
Matsunaga, S.	2004	No description of intervention
Matz, P.G.	2006	Review article
Mazanec, D.	2007	Inclusion of radiculopathy
Mochizuki, M.	2009	No description of intervention
Sahin, N.	2013	Abstract only
Sampath, P.	2000	Did not report outcomes of interest
Sanders, M.	1988	Analysis of lumbar condition
Sumi, M.	2012	Duplicate sample population (Shimomura,T.)
Wang, G.Q.	2014	Abstract only
Wang, Y.L.	1997	Not in English
Wu, J.C.	2012	Did not report outcomes of interest

SE Table B. Data Summary Table

Author	Follow-up (months)	Follow-up (%)	Pre-treatment score ± SD /range ( )	Post-treatment score	Baseline Characteristics
Fukui, K *et al* (1990)	30*	NR	11.1 (NR)	12.8 (NR)	N=50 Mean age= 58.6 years Male= 79%
Kadanka, Z. *et al* (2002;2011)	0 6 12 24 36 120	100 NR NR NR 83 78	14.6 (14.1-15.2) † --	 14.9 (14.3-15.6) 15.0 (14.4-15.6) 14.6 (14.1-15.2) 14.7 (14.0-15.3) 15‡ (12.2 -18.0)	N=35 Mean age= 54 Male=74.3%
Kong, L. *et al* (2013)	40*	87	Surgical §: 14.0 ±1.1 Conservative: 14.2 ±1.0	Surgical: 11.1 ±0.8 Conservative: 14.2 ±1.3	N=90 Mean age= 57.8 years Male= 58%
Li, F.N. *et al* (2014)	30.7*	NR	14.37	15.45	N=38 Mean age= 51.7 years Male= 52%
Matsumoto, M. *et al* (2001)	0 3 6 47*	NA	13.8 ±1.6 **	 14.2 ±1.4 14.3 ±1.3 16.1 ±0.9	N=27 Mean age= 44.4 years Male= 74%
Shimomura, T. *et al* (2007)	35.6*	80	14.6 ±1.3	14.7 ±2.0	N= 70 Mean age= 55.1 years Male= 54%

* Reported value is mean final follow-up time (mo)

† Modified JOA (mJOA) out of 18.0 points total

‡ Reported as median JOA score at final follow-up

§ Both groups underwent the same conservative treatment initially; Surgical group (n=21) assigned to surgical intervention after deterioration of condition (mean 2.9 point reduction in JOA). The remaining 19 subjects (conservative group) continued with conservative treatment until final follow-up (mean of 40 months).

** Two groups combined with weighted mean values

Modified Newcastle-Ottawa Scale (NOS)

0 = Definitely no (high risk of bias)
1 = Mostly no
2 = Mostly yes
3 = Definitely yes (low risk of bias)

	Selection Bias	**Performance Bias**	**Detection Bias**		**Information Bias**		
	Appropriate and representative source population?	Adequate Sample size?	Appropriate statistical methods?	Little missing data?	Explicitly stated and appropriate outcome measurement?	Objective assessment of outcome?	**Risk of bias**
Fukui *et al* 1990	2	2	2	0	3	3	**Moderately Low Risk of Bias**
Kadanka *et al* 2002, 2011	3	1	2	1	3	3	**Moderately Low Risk of Bias**
Kong *et al* 2013	3	3	3	2	3	3	**Moderately Low Risk of Bias**
Li *et al* 2014	1	1	2	0	3	3	**Moderately High Risk of Bias**
Matsumoto *et al* 2001	2	0	3	0	3	3	**Moderately High Risk of Bias**
Nakamura *et al* 1998	0	3	3	2	3	3	**Moderately Low Risk of Bias**
Shimomura *et al* 2007	2	3	3	2	3	3	**Moderately Low Risk of Bias**
Yoshimatsu *et al* 2001	2	2	3	0	3	3	**Moderately Low Risk of Bias**

Domain of evaluation: Methods for selecting study participants (i.e. Selection bias)

Is the source population (cases, controls, cohorts) appropriate and representative of the population of interest?

Example of low risk of bias: A consecutive sample or random selection from a population that is representative of the condition under study.

Example of moderate risk of bias: A consecutive sample or random selection from a population that is not highly representative of the condition under study.

Example of high risk of bias: The source population cannot be defined or enumerated (i.e. volunteering or self-recruitment).

Domain of evaluation: Methods to control confounding (i.e. Performance bias)

Is the sample size adequate and is there sufficient power to detect a meaningful difference in the outcome of interest?

Example of low risk of bias: Sample size was adequate and there was sufficient power to detect a difference in the outcome.

Example of high risk of bias: Sample size was small and there was not enough power to test outcome of interest.

Did the study identify and adjust for any variables or confounders that may influence the outcome?

Example of low risk of bias: The study identified and adjusted for all possible confounders that may influence estimates of association between exposure and outcome (i.e. Was the patient being treated for a medical condition such as chronic pain and was being prescribed opioids while on methadone treatment?)

Example of moderate risk of bias: The study identified and reported possible variables that may influence the outcome but did not explore the interaction.

Example of high risk of bias: The study either did not report any variables of influence or acknowledge variables of influence when it was clear they were present.

Domain of evaluation: Statistical methods (i.e. Detection bias)

Did the study use appropriate statistical analysis methods relative to the outcome of interest?

Example of low risk of bias: The study reported use of appropriate statistical analysis as required (i.e. adjusting for an unbalanced distribution of a specific covariate among sexes, or correcting for multiple testing error)

Example of moderate risk of bias: The study either used correct statistical methods but did not report them well, or used incorrect methods but reported them in detail.

Example of high risk of bias: The study did not use appropriate statistical analysis as required (i.e. did not adjust for an unbalanced distribution of a specific covariate among sexes, or correct for multiple testing error when necessary) or did not report them adequately.

Is there little missing data and did the study handle it accordingly?

Example of low risk of bias: The study acknowledged missing data to be less than 10% and specified the method used to handle it.

Example of moderate risk of bias: The study had greater than 15% missing data but specified the method used to handle it.

Example of high risk of bias: The study had greater than 15% missing data and did not handle it at all.

Domain of evaluation: Methods for measuring outcome variables (i.e. Information bias)

Is the methodology of the outcome measurement appropriate and explicitly stated?

Example of low risk of bias: The study provided a detailed description of the outcome measure(s) which are appropriate for the outcome of interest.

Example of moderate risk of bias: The study provided a somewhat complete description of outcome measurements and they are justified.

Example of high risk of bias: The study provided limited information on the methods of measuring the outcome and the measure is not appropriate considering the outcome.

Is there an objective assessment of the outcome of interest?

Example of low risk of bias: The study used objective methods to discern the outcome of participants (i.e. laboratory measurements, medical records).

Example of moderate risk of bias: The study relied on subjective data as the primary method to discern outcome of participants (i.e. self-report).

Example of high risk of bias: The study had limited reporting about outcome assessment.

SE Table D. GRADE Evaluation Details, Clinical Question 1

Clinical Question 1: What is the change in function, pain and quality of life following structured nonoperative treatment?								Effect Size
Outcome	**Sample Size**	**Risk of bias**	**Inconsistency**	**Indirectness**	**Imprecision**	**Publication bias**	**Overall quality of evidence**	**% or mean difference (MD)**
mJOA/JOA improvement	1 prospective cohort (n=50)*	Serious risk of bias†	Inconsistency unknown	No serious indirectness	Serious risk of imprecision	Undetected	INSUFFICIENT	MD: 1.7
	3 prospective cohorts (n=195)	Serious risk of bias†	Serious risk of inconsistency‡	No serious indirectness	No serious risk of imprecision	Undetected	INSUFFICIENT§	Range of MDs: 0 to 0.1
	3 retrospective cohorts (n=129)	Serious risk of bias†	Serious risk of inconsistency‡	No serious indirectness	Serious risk of imprecision	Undetected	INSUFFICIENT§	Range of MDs: 1.1 to 2.3
% patients with JOA improvement**	1 retrospective cohort (n=57)	Serious risk of bias†	Inconsistency unknown	No serious indirectness	Serious risk of imprecision	Undetected	INSUFFICIENT	18% (15/57)

* One study using mJOA scale

† Serious risk of bias: the majority of studies did not meet two or more important criteria of a good quality RCT or cohort

‡ Serious inconsistency: point estimates vary across studies in such a way that affects the confidence of the effect estimate

§ Downgraded 1 due to risk of bias and 1 for inconsistency

** Threshold for JOA improvement not defined

SE Table E: GRADE Evaluation Details, Clinical Question 2

Clinical Question 2: Does the change in function, pain and quality of life vary depending on treatment type?								Treatment groups		Effect Size
Outcome	**Sample Size**	**Risk of bias**	**Inconsistency**	**Indirectness**	**Imprecision**	**Publication bias**	**Overall quality of evidence**	**Rigorous (%)**	**Nonrigorous (%)**	**Relative Risk**
% patients with JOA improvement‡	1 retrospective cohort (n=57)	Serious risk of bias*	Inconsistency unknown	No serious indirectness	Serious risk of imprecision	Undetected	INSUFFICIENT	38 (14/37)	6 (2/32)	6.1 (95% CI 1.5, 24.6)

* Serious risk of bias: the majority of studies did not meet two or more important criteria of a good quality RCT or cohort

† Serious inconsistency: point estimates vary across studies in such a way that affects the confidence of the effect estimate

‡ Threshold for JOA improvement not defined

SE Table F. GRADE Evaluation Details, Clinical Question 4

Clinical Question 4: What are the negative patient outcomes and harms?								Effect Size
Outcome	**Sample Size**	**Risk of bias**	**Inconsistency**	**Indirectness**	**Imprecision**	**Publication bias**	**Overall quality of evidence**	**% difference**
Surgery following nonoperative care	2 prospective cohorts, 3 retrospective cohorts (n=288)	Serious risk of bias*	Serious risk of inconsistency†	No serious indirectness	Serious risk of imprecision	Undetected	INSUFFICIENT‡	Range: 23 – 54%

* Serious risk of bias: the majority of studies did not meet two or more important criteria of a good quality RCT or cohort

† Serious inconsistency: point estimates vary across studies in such a way that affects the confidence of the effect estimate

‡ Downgraded 1 due to risk of bias and 1 for inconsistency